Anthony Jude Di Were Omolo from University of Nairobi, Nairobi (Kenya) to go to Manchester Royal Infirmary, Manchester (UK) with R. Gokal, M.D.

An 8th fellow will be trained in the U.K. and supported by the National Kidney Research Fund from U.K.:

Peter Dawari Hart from University College Hospital, Oyo State Ibadan (Nigeria) to go to The London Hospital Whitechapel, London (UK) with F. P. Marsh, F.R.C.P.

Erratum

In the recent article by N. Topley et al, *Kidney Int* 36: 609–616, 1989, an error appeared in the first sentence of the abstract. The word, independent, should read dependent. The abstract is reprinted in its corrected entirety below.

Type 1 fimbriate strains of *escherichia coli* initiate renal parenchymal scarring. The renal scarring which characterizes chronic pyelonephritis is initiated by bacterial infection and is dependent on the activation of an inflammatory response. Although the presence of polymorphonuclear leukocytes (PMN) is essential for initiating the scarring process, the bacterial structures responsible for their activation have not been investigated. In an animal model of chronic pyelonephritis the surface area of the renal scars produced by Type 1 fimbriate *escherichia coli* (*E. coli*) was significantly greater than that of those produced by P fimbriate and non-fimbriate strains ($P < 0.01$). The activation of human PMN by the same Type 1 fimbriate organisms resulted in a significant release of lysosomal neutral protease activity ($P < 0.001$) and activation of the respiratory burst ($P < 0.01$). The neutral protease release in response to P fimbriate and non-fimbriate organisms was not significantly increased. The extent of renal scarring also correlated with the release of neutral protease activity ($P < 0.02$) and with the degree of activation of the respiratory burst ($P < 0.05$). These results demonstrate that the ability of *E. coli* strains to cause renal scars may be related to their capacity to express Type 1 fimbria, which may be a causative factor in the in vivo activation of the inflammatory response.

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Thomas E. Andreoli, M.D.
Editor, *Kidney International*
Department of Medicine
University of Arkansas College of Medicine
4301 West Markham
Slot #712
Little Rock, Arkansas 72205 USA
Telephone numbers for the Editorial Office are:
(501) 686-9609
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